5180981

FILE 'HOME' ENTERED AT 16:51:45 ON 20 JAN 2003

=> file biosis

=> s antitrypsin/ti or anti trypsin/ti
2318 ANTITRYPSIN/TI
146884 ANTI/TI
10710 TRYPSIN/TI
1629 ANTI TRYPSIN/TI
((ANTI(W)TRYPSIN)/TI)
L1 3947 ANTITRYPSIN/TI OR ANTI TRYPSIN/TI

=> s hiv/ti or immunodeficiency/ti
74121 HIV/TI
28535 IMMUNODEFICIENCY/TI
L2 99459 HIV/TI OR IMMUNODEFICIENCY/TI

=> s l1 and l2
L3 7 L1 AND L2

=> d bib ab 1-7

L3 ANSWER 1 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2002:93434 BIOSIS
DN PREV200200093434
TI Inhibition of ***HIV*** -2ROD replication in a lymphoblastoid cell line by the alpha1- ***antitrypsin*** Portland variant (alpha1-PDX) and the decRVKRcmk peptide: Comparison with ***HIV*** -1LAI.
AU Bahbouhi, Bouchaib; Bendjennat, Mourad; Chiva, Cristina; Kogan, Marcelo; Albericio, Fernando; Giralt, Ernest; Seidah, Nabil Georges; Bahraoui, Elmostafa (1)
CS (1) Laboratoire d'Immuno-Virologie, Universite Paul Sabatier, UFR/SVT, 118, Route de Narbonne, Bat 4R3, 31062, Toulouse Cedex: bahraoui@cict.fr France
SO Microbes and Infection, (November, 2001) Vol. 3, No. 13, pp. 1073-1084. print.
ISSN: 1286-4579.
DT Article
LA English

AB We investigated the effects of alpha1-antitrypsine Portland variant (alpha1-PDX) and decanoylRVKRchloromethylketone (decRVKRcmk) on HIV-2ROD replication in the Jurkat lymphoblastoid cell line. To this end, cells were stably transfected with the alpha1-PDX (J-PDX) and used as targets for HIV-2ROD infection. Controls were prepared with the empty vector (J-pcDNA3). HIV-2ROD and HIV-1LAI replications were significantly inhibited and delayed in the presence of the alpha1-PDX protein. When decRVKRcmk was used at 35 muM, inhibition rates were 70-80% for HIV-2ROD and HIV-1LAI, while total inhibition occurred at 70 muM. Control peptides consisting of decanoylRVKR and acetylYVADcmk had no effect. In the presence of the alpha1-PDX or the decRVKRcmk at 35 muM, the infectivity of HIV-2ROD and HIV-1LAI produced was 3-4-fold lower. Both molecules inhibited syncytium formation by HIV-2ROD and HIV-1LAI to a considerable extent. Finally, the inhibition of viral replication was correlated with the ability of the decRVKRcmk at 35 and 70 muM and of the alpha1-PDX, to inhibit the processing of envelope glycoprotein precursors. The alpha1-PDX protein and the decRVKRcmk peptide at 35 muM inhibited HIV-2 and HIV-1 to a similar level suggesting that identical or closely related endoproteases are involved in the maturation of their envelope glycoprotein precursors into surface and transmembrane glycoproteins. The significant inhibition observed with alpha1-PDX indicates that furin or furin-like endoproteases appear to play a major role in the maturation process.

L3 ANSWER 2 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2002:1005 BIOSIS
DN PREV200200001005
TI Replication of ***HIV*** -1 viruses in the presence of the Portland alpha1- ***antitrypsin*** variant (alpha1-PDX) inhibitor.
AU Bahbouhi, Bouchaib; Seidah, Nabil Georges; Bahraoui, Elmostafa (1)
CS (1) Laboratoire d'immuno-virologie, EA 30-38 UFR/SVT, Universite Paul Sabatier, 31062, Toulouse: bahraoui@cict.fr France
SO Biochemical Journal, (15 November, 2001) Vol. 360, No. 1, pp. 127-134. print.
ISSN: 0264-6021.
DT Article
LA English
AB The Portland alpha1-antitrypsin variant (alpha1-PDX) inhibits gp160 cleavage into gp120 and gp41 by different prohormone convertases (PCs) including furin, PC5 and PC7. Jurkat cells stably transfected with this inhibitor (J-PDX cells) and, as controls, Jurkat cells transfected with the empty vector (J-pcDNA3) were tested for their susceptibility to HIV-1 infection. We found that HIV-1 replication was significantly impaired in J-PDX cells. However, the analysis of the infectivity of HIV-1 viruses

produced in J-PDX cells on different days during the infection indicated that they recovered infectivity starting from 13 days post-infection. The sequencing of viruses collected earlier and later from J-PDX cells revealed no mutations in envelope-glycoprotein precursor (Env) maturation sites or in the N-terminal sequence of gp41 fusion peptide, which plays a key role in membrane fusion. Although conserved mutations were detected at the C-terminus of the gp41 fusion peptide and ectodomain, the replication of mutant HIV-1 viruses produced on day 20 in J-PDX cells was inhibited at a similar level to wild-type viruses after a second passage in J-PDX cells. We then investigated the expression of the alpha1-PDX protein, and found that HIV-1 replication activated its proteolysis since the 54 kDa cleaved form became predominant later on in the infection. In contrast, the expression of PC7, a protein that is transported through the secretory pathway, was unaltered in HIV-1 infected cells. We conclude that recovered HIV-1 infectivity in J-PDX cells was due to a loss of alpha1-PDX activity via its extensive processing by intracellular proteases that cleave it through the substrate pathway.

L3 ANSWER 3 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2001:84306 BIOSIS
DN PREV200100084306
TI Alpha-1- ***antitrypsin*** inhibits human ***immunodeficiency*** virus type 1.
AU Shapiro, Leland (1); Pott, Gregory B.; Ralston, Annemarie H.
CS (1) Department of Medicine, Division of Infectious Diseases, University of Colorado Health Sciences Center, Denver, CO, 80262: leland.shapiro@uchsc.edu USA
SO FASEB Journal, (January, 2001) Vol. 15, No. 1, pp. 115-122. print. ISSN: 0892-6638.
DT Article
LA English
SL English
AB Several observations suggest the existence of potent endogenous suppressors of human immunodeficiency virus type 1 (HIV-1) production, and inhibitors of serine proteases may participate in this effect. Alpha-1-antitrypsin (AAT) is the most abundant circulating serine protease inhibitor. Physiological AAT concentrations inhibited HIV-1 production in chronically infected U1 monocytic cells, reduced virus replication in freshly infected peripheral blood mononuclear cells, and blocked infection of permissive HeLa cells. In U1 cells, AAT suppressed activation of the HIV-1-inducing transcription factor NF-kappaB. Similar results were obtained using CE-2072, a synthetic inhibitor of host serine proteases. HIV-1 did not replicate in blood obtained from healthy volunteers, but

marked replication was observed in blood from individuals with hereditary AAT deficiency. These results identify AAT as a candidate circulating HIV-1 inhibitor in vivo. Two different mechanisms of AAT-induced HIV-1 inhibition were identified, including reduced HIV-1 infectivity and blockade of HIV-1 production. A novel host-pathogen interaction is suggested, and an alternative strategy to treat HIV-1-related disease may be possible.

L3 ANSWER 4 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2001:41274 BIOSIS
DN PREV200100041274
TI Effect of alpha-1 ***antitrypsin*** Portland variant (alpha1-PDX) on ***HIV*** -1 replication.
AU Bahbouhi, Bouchaib; Bendjennat, Mourad; Guetard, Denise; Seidah, Nabil Georges; Bahraoui, Elmostafa (1)
CS (1) Laboratoire d'Immuno-virologie, Universite Paul Sabatier, UFR/SVT, 31062, Toulouse: bahraoui@cict.fr France
SO Biochemical Journal, (15 November, 2000) Vol. 352, No. 1, pp. 91-98. print.
ISSN: 0264-6021.
DT Article
LA English
SL English
AB The present work investigated the potential role of alpha-1 antitrypsin Portland variant (alpha1-PDX), a bioengineered serine proteinase inhibitor (serpin), in the interference with the viral replication of HIV-1, induction of syncytia and maturation of envelope glycoprotein gp160 to gp120 and gp41. A Jurkat lymphoid cell line transfected with a plasmid containing the alpha1-PDX cDNA (J-PDX) and expressing the protein in a stable manner was infected with HIV-1Lai. Controls were Jurkat cells transfected with the same vector pcDNA3 without the cDNA insert (J-pcDNA3). The results showed that viral replication of HIV-1 was significantly inhibited with a delay in replication kinetics in J-PDX cells as compared with J-pcDNA3 cells. In addition, a comparison of the infectious capacity of viruses produced in the presence and absence of alpha1-PDX revealed that this capacity differed. It was found that alpha1-PDX exerts its effect by interfering with the formation of syncytia between J-PDX cells infected with gp160 recombinant vaccinia virus, or after infection by HIV-1 and co-culture with uninfected Molt-4 cells. In contrast, when the same experiments were performed with J-pcDNA3 cells, a large number of syncytia was obtained. Analysis of viral proteins by Western blotting and densitometry showed that the inhibition of the cytopathic effect of HIV-1 and viral replication was correlated with the

capacity of alpha1-PDX to interfere with the maturation of gp160 to gp120 and gp41.

L3 ANSWER 5 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1996:125381 BIOSIS
DN PREV199698697516
TI Faecal alpha-1 ***antitrypsin*** as a marker of gastrointestinal disease in ***HIV*** antibody positive individuals.
AU Sharpstone, D.; Rowbottom, A.; Nelson, M.; Gazzard, B. (1)
CS (1) Kobler Centre, Chelsea Westminster Hosp., 369 Fulham Road, London SW10 9TH UK
SO Gut, (1996) Vol. 38, No. 2, pp. 206-210.
ISSN: 0017-5749.
DT Article
LA English
AB Hypoalbuminaemia and diarrhoea are common complications of HIV infection and substantial causes of morbidity, but the specific intestinal pathologies that cause enteric protein loss have not been clearly defined. Two hundred and twenty stool samples from patients with a variety of HIV related conditions were analysed for faecal alpha-1 antitrypsin. Patients with intestinal Kaposi's sarcoma had a significantly raised faecal alpha-1 antitrypsin value and hypoalbuminaemia. A faecal alpha-1, antitrypsin value of greater than 0.3 mg/g wet stool has a sensitivity of 94% and a specificity of 76% for the diagnosis of intestinal Kaposi's sarcoma in HIV positive individuals. Patients with cytomegalovirus and bacterial enteritis had raised faecal a, antitrypsin values but levels were normal for all other intestinal pathologies compared with pathogen negative stool. The combination of faecal alpha-1 antitrypsin concentration greater than 0.2 mg/g, a negative stool culture for enteric bacteria, and the absence of palatal Kaposi's sarcoma has a sensitivity of 55% and specificity of 88% for the diagnosis of enteric cytomegalovirus infection.

L3 ANSWER 6 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1994:31906 BIOSIS
DN PREV199497044906
TI Inhibition of ***HIV*** -1 gp160-dependent membrane fusion by a furin-directed alpha-1- ***antitrypsin*** variant.
AU Anderson, Eric D.; Thomas, Laurel; Hayflick, Joel S.; Thomas, Gary (1)
CS (1) Vollum Inst., Oregon Health Sci. University, Portland, OR 97201 USA
SO Journal of Biological Chemistry, (1993) Vol. 268, No. 33, pp. 24887-24891.
ISSN: 0021-9258.
DT Article
LA English

AB Furin is a membrane-associated calcium-dependent serine endoprotease that cleaves proproteins on the carboxyl side of the consensus sequence -Arg-X-Lys/Arg-Arg-. Using site-directed mutagenesis, a variant alpha-1-antitrypsin (alpha-1-AT) was constructed which contains in its reactive site -Arg-X-X-Arg-, the minimal sequence required for efficient processing by furin (Molloy, S. S., Bresnahan, P. A., Leppla, S. H., Klimpel, K. R., and Thomas, G. (1992) J. Biol. Chem. 267,16396-16402). This alpha-1-AT variant, (Arg-355Arg-358)alpha-1-AT (alpha-1-PDX), is greater than 3,000-fold more effective than (Arg-358)alpha-1-AT (alpha-1-AT Pittsburgh, alpha-1-PIT) at inhibiting furin in vitro (K-0.5 = 0.03 mu-g/ml). Furthermore, the P4 Arg in alpha-1-PDX greatly attenuates the thrombin inhibitory properties of this serpin (gt 300-fold) compared with alpha-1-PIT (which contains a P4 Ala), thus increasing the selectivity of alpha-1-PDX for furin. Expression studies show that alpha-1-PDX, and not alpha-1-PIT, blocks the processing of two furin substrates, pro-beta-nerve growth factor and human immunodeficiency virus (HIV)-1 gp160 in transfected cells. In addition, a syncytium assay shows that alpha-1-PDX blocks the membrane fusogenic properties of HIV-1 gp160. The potential use of alpha-1-PDX in manipulating the activation of proproteins in a tissue- and time-specific manner is discussed.

L3 ANSWER 7 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1987:435390 BIOSIS
DN BR33:94217
TI APPLICATIONS OF PROTEIN ENGINEERING THE EXAMPLES OF HUMAN ALPHA-1
ANTITRYPSIN AND ***HIV*** ENV PROTEIN.
AU LECOCQ J M; KIENY M P; COURTNEY M
CS TRANSGENE SA, 11 RUE DE MOLSHEIM, 6700 STRASBOURG CEDEX, FR.
SO PROTEIN ENGINEERING '87, OXFORD, ENGLAND, UK, APRIL 5-8, 1987. PROTEIN ENG. (1987) 1 (3), 237-238.
CODEN: PRENE9. ISSN: 0269-2139.
DT Conference
FS BR; OLD
LA English

=> log hold

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		16.97	17.18

SESSION WILL BE HELD FOR 60 MINUTES

510048 C

FILE 'HOME' ENTERED AT 15:03:33 ON 20 JAN 2003

=> file medline

=> s antitrypsin or anti trypsin
10136 ANTITRYPSIN
383524 ANTI
55440 TRYPSIN
165 ANTI TRYPSIN
(ANTI(W)TRYPSIN)
L1 10223 ANTITRYPSIN OR ANTI TRYPSIN

=> s hib
L2 1288 HIB

=> s hiv
L3 122871 HIV

=> s l1(10a)l3
L4 9 L1(10A)L3

=> d bib kwic 1-9

L4 ANSWER 1 OF 9 MEDLINE
AN 2003021653 IN-PROCESS
DN 22415031 PubMed ID: 12527499
TI Enteric dysfunction in patients with asymptomatic HIV-infection versus patients with hypoproteinemia/hypalbuminemia and advanced HIV-infection.
AU Wasmuth J-C; Schliefer K; Scheurlen C; Sudhop T; Rockstroh J K; Sauerbruch T; Spengler U
CS Department of Internal Medicine I, University of Bonn, Germany.. j-c.wasmuth@uni-bonn.de
SO EUROPEAN JOURNAL OF MEDICAL RESEARCH, (2002 Dec 17) 7 (12) 536-42.
Journal code: 9517857. ISSN: 0949-2321.

CY Germany: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20030117
Last Updated on STN: 20030117
AB BACKGROUND: It is unclear whether enteric dysfunction and protein losses contribute to hypoproteinemia, which heralds poor survival in ***HIV*** infection. METHODS: We investigated alpha-1- ***antitrypsin*** -clearance (AAT-CL), D-xylose resorption and total gut transition time in 14 HIV+ patients with hypoproteinemia (serum protein < 6 g/dl, albumin. .
.

L4 ANSWER 2 OF 9 MEDLINE
AN 2001688814 MEDLINE
DN 21566376 PubMed ID: 11709287
TI Inhibition of HIV-2(ROD) replication in a lymphoblastoid cell line by the alpha1- ***antitrypsin*** Portland variant (alpha1-PDX) and the decRVKRcmk peptide: comparison with ***HIV*** -1(LAI).
AU Bahbouhi B; Bendjennat M; Chiva C; Kogan M; Albericio F; Giralt E; Seidah N G; Bahraoui E
CS Laboratoire dimmuno-virologie, Universite Paul Sabatier, Bat 4R3, UFR/SVT, 118, route de Narbonne, 31062 cedex, Toulouse, France.. bahraoui@cict.fr
SO Microbes Infect, (2001 Nov) 3 (13) 1073-84.
Journal code: 100883508. ISSN: 1286-4579.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200207
ED Entered STN: 20011210
Last Updated on STN: 20020724
Entered Medline: 20020723
TI Inhibition of HIV-2(ROD) replication in a lymphoblastoid cell line by the alpha1- ***antitrypsin*** Portland variant (alpha1-PDX) and the decRVKRcmk peptide: comparison with ***HIV*** -1(LAI).

L4 ANSWER 3 OF 9 MEDLINE

AN 2001643959 MEDLINE
DN 21552901 PubMed ID: 11695999
TI Replication of ***HIV*** -1 viruses in the presence of the Portland alpha1- ***antitrypsin*** variant (alpha1-PDX) inhibitor.
AU Bahbouhi B; Seidah N G; Bahraoui E
CS Laboratoire d'immuno-virologie, EA 30-38 Universite Paul Sabatier, UFR/SVT, 31062 Toulouse, France.
SO BIOCHEMICAL JOURNAL, (2001 Nov 15) 360 (Pt 1) 127-34.
Journal code: 2984726R. ISSN: 0264-6021.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200112
ED Entered STN: 20011107
Last Updated on STN: 20020123
Entered Medline: 20011218
TI Replication of ***HIV*** -1 viruses in the presence of the Portland alpha1- ***antitrypsin*** variant (alpha1-PDX) inhibitor.

L4 ANSWER 4 OF 9 MEDLINE
AN 2001485554 MEDLINE
DN 21418914 PubMed ID: 11527807
TI Self antigen prognostic for human immunodeficiency virus disease progression.
AU Bristow C L; Patel H; Arnold R R
CS Department of Pathology and Laboratory Medicine, University of North Carolina-Chapel Hill, Chapel Hill, NC 27514, USA..
bristoc@mail.rockefeller.edu
NC DE-08671 (NIDCR)
DE-11190 (NIDCR)
HD-37260 (NICHD)
SO CLINICAL AND DIAGNOSTIC LABORATORY IMMUNOLOGY, (2001 Sep) 8 (5) 937-42.
Journal code: 9421292. ISSN: 1071-412X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 200112
ED Entered STN: 20010903
Last Updated on STN: 20020122
Entered Medline: 20011204
AB We have recently found that an extracellular protein, alpha(1) proteinase inhibitor (alpha(1)PI; alpha(1) ***antitrypsin***), is required for in vitro human immunodeficiency virus (***HIV***) infectivity outcome. We show here in a study of HIV-seropositive patients that decreased viral load is significantly correlated with decreased. . .

L4 ANSWER 5 OF 9 MEDLINE
AN 2001485553 MEDLINE
DN 21418913 PubMed ID: 11527806
TI Slow human immunodeficiency virus (HIV) infectivity correlated with low HIV coreceptor levels.
AU Bristow C L
CS Department of Pathology and Laboratory Medicine, University of North Carolina-Chapel Hill, Chapel Hill, NC 27514, USA..
bristoc@mail.rockefeller.edu
SO CLINICAL AND DIAGNOSTIC LABORATORY IMMUNOLOGY, (2001 Sep) 8 (5) 932-6.
Journal code: 9421292. ISSN: 1071-412X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200112
ED Entered STN: 20010903
Last Updated on STN: 20020122
Entered Medline: 20011204
AB . . . (HIV) infectivity outcome. It has recently been shown that human leukocyte elastase (HLE) and its ligand alpha(1) proteinase inhibitor (alpha(1)PI; alpha(1) ***antitrypsin***) act as ***HIV*** fusion cofactors. The present study shows that decreased HIV infectivity is significantly correlated with decreased cell surface density of HLE. . .

L4 ANSWER 6 OF 9 MEDLINE

AN 2001092112 MEDLINE
DN 20517259 PubMed ID: 11062061
TI Effect of alpha-1 ***antitrypsin*** Portland variant (alpha 1-PDX) on ***HIV*** -1 replication.
AU Bahbouhi B; Bendjennat M; Guetard D; Seidah N G; Bahraoui E
CS Laboratoire d'immuno-virologie, Universite Paul Sabatier, UFR/SVT, 31062 Toulouse, France.
SO BIOCHEMICAL JOURNAL, (2000 Nov 15) 352 Pt 1 91-8.
Journal code: 2984726R. ISSN: 0264-6021.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200101
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010125
TI Effect of alpha-1 ***antitrypsin*** Portland variant (alpha 1-PDX) on ***HIV*** -1 replication.

L4 ANSWER 7 OF 9 MEDLINE
AN 1999295776 MEDLINE
DN 99295776 PubMed ID: 10369102
TI The macrophage origin of the HIV-expressing multinucleated giant cells in hyperplastic tonsils and adenoids.
AU Orenstein J M; Wahl S M
CS Department of Pathology, George Washington University Medical Center, Washington, DC 20037, USA.. patjmo@gwumc.edu
NC DE 12585 (NIDCR)
SO ULTRASTRUCTURAL PATHOLOGY, (1999 Mar-Apr) 23 (2) 79-91.
Journal code: 8002867. ISSN: 0191-3123.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; AIDS
EM 199907
ED Entered STN: 19990806
Last Updated on STN: 20000303

Entered Medline: 19990729

AB . . . hybridization (ISH), light and transmission electron microscopy (TEM), and immunohistochemistry (IHC) (HIV Gag p24 protein, S-100, p55, CD68, HAM56, lysozyme, alpha-1- ***anti*** - ***trypsin*** , and alpha-1-anti-chymotrypsin). In ***HIV*** + pediatric and adult surgical specimens (n = 11), the giant cells and their mononuclear counterpart were positive for both macrophage. . .

L4 ANSWER 8 OF 9 MEDLINE

AN 1998025764 MEDLINE

DN 98025764 PubMed ID: 9382053

TI Mucosal injury and disruption of intestinal barrier function in HIV-infected individuals with and without diarrhea and cryptosporidiosis in northeast Brazil.

AU Lima A A; Silva T M; Gifoni A M; Barrett L J; McAuliffe I T; Bao Y; Fox J W; Fedorko D P; Guerrant R L

CS Clinical Research Unit, University Hospital, Federal University of Ceara, Fortaleza, Brazil.

NC PO1-AI-26512 (NIAID)

SO AMERICAN JOURNAL OF GASTROENTEROLOGY, (1997 Oct) 92 (10) 1861-6.

Journal code: 0421030. ISSN: 0002-9270.

Report No.: PIP-128564; POP-00270095.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; Population; AIDS

EM 199711

ED Entered STN: 19971224

Last Updated on STN: 20021101

Entered Medline: 19971112

AB . . . (p = 0.02). One patient with microsporidial infection had a nearly 3-fold higher L:M ratio than controls without diarrhea. Alpha 1- ***Antitrypsin*** was positive in 40% of ***HIV*** -positive patients with cryptosporidial infections and none of 12 HIV-positive patients with non-cryptosporidial diarrhea. Fecal lactoferrin or leukocytes were increased in. . . lactulose excretion (mucosal barrier disruption). The single patient with microsporidial infection had a nearly 3-fold higher

ratio than healthy volunteers. Alpha1- ***antitrypsin*** tests were positive in two of five (40%) ***HIV*** -positive patients with cryptosporidial infections compared with none of 12 HIV-infected patients with non-cryptosporidial diarrhea. These findings confirm that HIV infection. . .

L4 ANSWER 9 OF 9 MEDLINE
AN 96246358 MEDLINE
DN 96246358 PubMed ID: 8801198
TI Faecal alpha 1 ***antitrypsin*** as a marker of gastrointestinal disease in ***HIV*** antibody positive individuals.
AU Sharpstone D; Rowbottom A; Nelson M; Gazzard B
CS Department of HIV/GUM, Chelsea and Westminster Hospital, London.
SO GUT, (1996 Feb) 38 (2) 206-10.
Journal code: 2985108R. ISSN: 0017-5749.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; AIDS
EM 199610
ED Entered STN: 19961015
Last Updated on STN: 19980206
Entered Medline: 19961001
TI Faecal alpha 1 ***antitrypsin*** as a marker of gastrointestinal disease in ***HIV*** antibody positive individuals.
AB . . . enteric protein loss have not been clearly defined. Two hundred and twenty stool samples from patients with a variety of ***HIV*** related conditions were analysed for faecal alpha 1 ***antitrypsin*** . Patients with intestinal Kaposi's sarcoma had a significantly raised faecal alpha 1 antitrypsin value and hypoalbuminaemia. A faecal alpha 1. . .

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